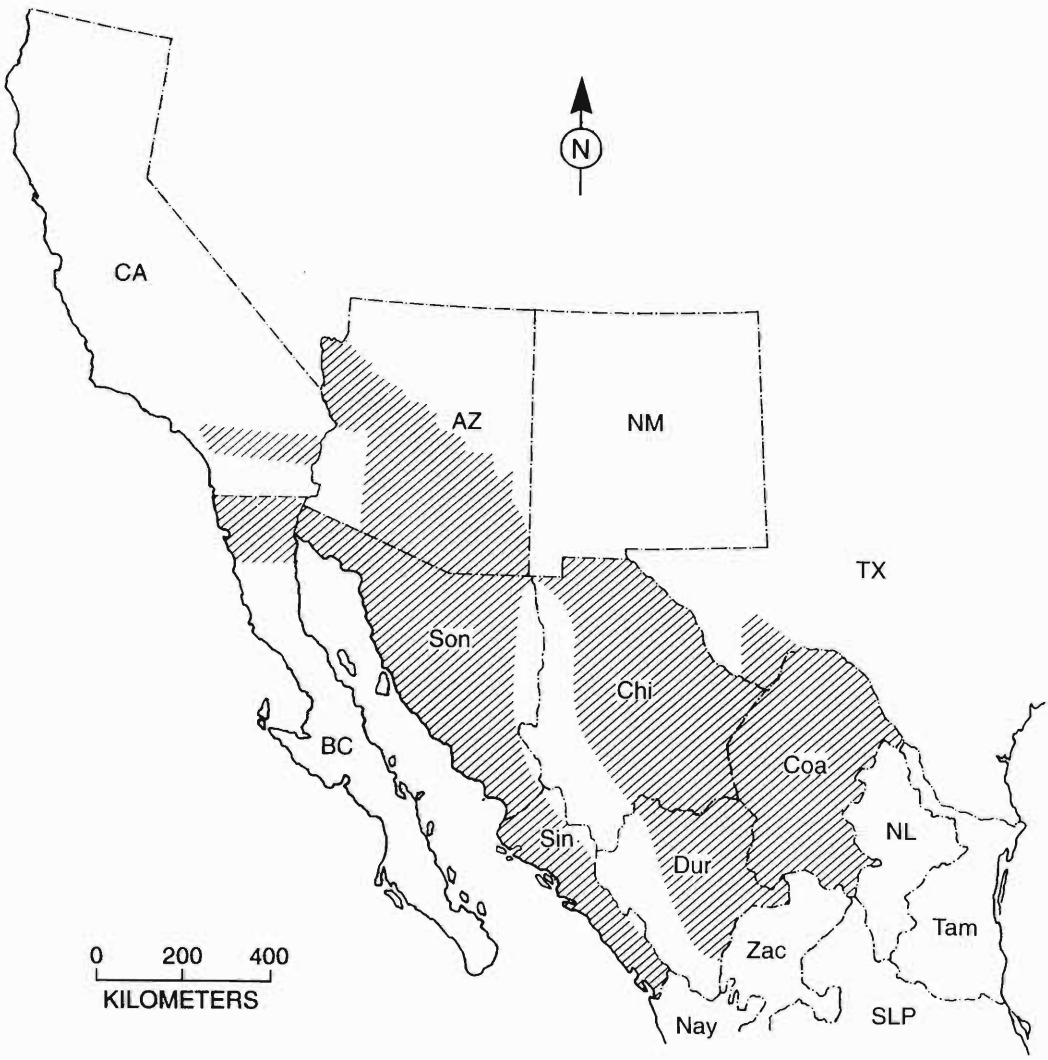

Figure 1. Map of Southwestern North America. Shaded sectors represent areas from which *Rhinocheilus lecontei* with oligacanthorhynchid cystacanths were found. Mexico: BC = Baja California; Coa = Coahuila; Chi = Chihuahua; Dur = Durango; Nay = Nayarit; NL = Nuevo Leon; Sin = Sinaloa; SLP = San Luis Potosí; Son = Sonora; Tam = Tamaulipas; Zac = Zacatecas.

snakes from Riverside County; in Texas, they were found only in snakes from Brewster County. No cystacanths were found in snakes from Nevada, (0/42), New Mexico (0/17), or Utah (0/1). The distribution pattern (Fig. 1) suggests that infected snakes are distributed primarily in Arizona and Mexico.

This is the fourth report of oligacanthorhynchid cystacanths from snakes of North America. Elkins and Nickol (1983) collected cystacanths from naturally infected *Agkistrodon piscivorous, Coluber constrictor, Lampropeltis getula, Nerodia cyclopion,* and *N. fasciata* from southern Louisiana and recovered cystacanths from experimentally infected *Thamnophis sirtalis, N. cyclopion,* and *N. fasciata.* Bolette (1997a) reported *Pachysentis canicola* from the western diamondback rattlesnake, *Crotalus atrox,* collected in Nolan County, Texas, and oligacanthorhynchid cystacanths from *Crotalus scutulatus* and *R. lecontei* collected in Maricopa County, Arizona (Bolette, 1997b).

For those acanthocephalans parasitic in terrestrial animals, the intermediate hosts are usually insects (Nickol, 1985). *Rhinocheilus lecontei* is known to eat insects (Stebbins, 1954) and thus might be expected to become infected. The significant difference in infection frequencies between adult and juvenile snakes may be a function of the number of insects eaten as well as the number of cystacanths in the insects eaten. Subsequent study will be required to more precisely determine the geographical distribution of the acanthocephalans found in this study; however, the absence of oligacanthorhynchid cystacanths in *R. lecontei* from Nevada, New Mexico, and much of California and Texas suggests an absence of definitive hosts in these areas.

We thank the following for permission to examine *Rhinocheilus lecontei:* Robert L. Bezy (Natural History Museum of Los Angeles County), Michael E. Douglas (Arizona State University), and Charles H. Lowe (University of Arizona). Chris H. Gardiner (Field Medical Service School, Camp Pendleton, California) confirmed the identification of larval acanthocephalans. The illustration was done by Peggy Firth.

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Research Note

The Effects of *Echinostoma trivolvis* Infection on the Fertility and Fecundity of Golden Hamsters (*Mesocricetus auratus*) and on the Infectivity of Their Progeny

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ABSTRACT: The effects of *Echinostoma trivolvis* infection on fertility and fecundity in the golden hamster (*Mesocricetus auratus*) was studied. The infectivity in the progeny from infected mothers was evaluated. The average litter size for infected hamsters was 8.4 ± 2.7 compared with 9.4 ± 4.0 ($P > 0.05$) from uninfected hamsters. Neonates from infected mothers showed decreased infectivity at ages 6, 7, and 8 wk postinfection. Neonates from uninfected mothers showed a greater infectivity when compared with animals born from infected mothers. The calculated percentage of resistance at 6, 7, and 8 wk of age was 48.9%, 69.3%, and 71.5%, respectively. Spleens from infected mothers showed a depletion of white pulp. The adrenal cortex from infected mothers was widened and composed predominately of lipid-poor reticularis-type cells.

KEY WORDS: *Echinostoma trivolvis*, fertility, fecundity, infectivity, *Mesocricetus auratus*.

Intestinal trematode infections are widespread in humans and animals. The factors that determine the innate resistance or susceptibility of a host to parasites are of considerable interest. Recent studies have centered on acquired resistance to echinostome infections in experimental rodent hosts. Little is known about the effects of helminths on their pregnant host. Bindseil and Hau (1991) showed that infection of BALB/cBOM mice with *Echinostoma caproni* had a negative influence on pregnancy; fewer fetuses were present in infected mice than in controls. Ovulation, fertilization, and egg implantation were not affected.

Pregnancy and lactation in the host do not appear to affect the course of cestode or trematode infections. Reproductive processes in the female host, however, increase susceptibility to infection with nematodes (Ogilvie and Jones, 1973).

Huffman and Fried (1990) suggested that a complex set of interrelating factors may govern the immune response in echinostome infections. The environment into which the young are born contains a myriad of infectious organisms from which the neonate must be protected. There are 2 routes whereby the neonate may gain protection from infectious organisms: via the placenta before birth and via colostrum and milk after birth (Paul, 1989). Both of these routes transfer maternal antibodies to the young (Carlier and Truyens, 1995).

The objectives of this study were to provide evidence for the effects of infection on fertility and fecundity in the golden hamster and to evaluate infectivity in the progeny from infected mothers. The relative adrenal and splenic weights and number of lymphatic nodules are reported from infected and noninfected animals. Parasite recovery, location, and dry weights are reported from infected animals.

Metacercarial cysts of *Echinostoma trivolvis* were obtained from the kidney and pericardial sac of laboratory-infected *Biomphalaria glabrata*. Outbred golden hamsters (*Mesocricetus auratus*) were obtained from the East Stroudsburg University Animal Care Facility. All animals were provided food and water ad libitum throughout the study.

Twenty-four female hamsters 20 wk of age were divided into 2 groups of 12 hamsters each. Group A was infected with 30 cysts of *E. trivolvis* per os, and group B animals were not infected. Fecal samples from group A hamsters were checked periodically to verify infection.

On day 52 postinfection (PI), all animals were bred after completion of 2 consecutive estrous cycles. Cyclicity was assessed by the presence of a postovulatory vaginal discharge on the morning after ovulation (Greenwald, 1962). Pregnancy was confirmed by obtaining a vaginal smear and staining with Papanicolau stain and identifying the characteristic cells present during

Table 1. Percentage of infectivity, relative splenic and adrenal weights, and number of lymphatic nodules for hamsters and mean worm dry weights.

Group*	Hamster age (wk)		% Infectivity	Mean (±SD) worm dry weight (mg)	Relative spleen weight ($\bar{x}$ ± SD)	Relative adrenal weight ($\bar{x}$ ± SD)	Mean (±SD) no. lymphatic nodules
	At infection	At necropsy					
A	20	36	39.7	1.70 ± 0.4	80.0 ± 5.0	35.0 ± 5.0	4.3 ± 1.9
B	20	36	0	0	118 ± 10.0	11.5 ± 5.0	3.6 ± 1.3
C	4	6	52.2	1.28 ± 0.1	100.3 ± 8.5	11.6 ± 3.2	3.6 ± 1.1
	5	7	67.6	2.09 ± 0.2	101.5 ± 19.6	12.8 ± 2.4	7.6 ± 1.2
	6	8	48.2	1.73 ± 0.1	85.9 ± 16.2	10.7 ± 3.7	6.8 ± 1.4
D	4	6	21.2	2.06 ± 0.6	108.6 ± 23.1	15.3 ± 3.3	5.3 ± 1.4
	5	7	20.7	1.50 ± 0.6	87.0 ± 20.8	15.3 ± 3.5	4.8 ± 0.7
	6	8	14.1	1.40 ± 0.4	100.0 ± 43.2	17.1 ± 0.8	5.3 ± 1.6

* A = infected mothers; B = uninfected controls; C = infected progeny from noninfected mothers; D = infected progeny from infected mothers.

pregnancy. The number, vigor, and average pup weight from both groups was recorded. Total pup weight was recorded daily for 2 wk postpartum.

The offspring from groups A and B were divided into 2 groups: group C consisted of 24 animals from uninfected mothers, and group D consisted of 24 animals from infected mothers. Sixteen juvenile hamsters from group C or group D were administered 15 cysts of *E. trivolvis* at 4, 5, or 6 wk of age. Eight hamsters from each group and from each age category were necropsied on day 14 PI. The hamsters from groups A and B were necropsied 16 wk PI. The small intestine was removed, and the serosal lymphatic nodules were counted. The intestine was opened, and the number of parasites was recorded and the percentage of infectivity was determined as follows:

$$\frac{\text{(number of parasites recovered)}}{\text{(number of cysts administered)}} \times 100.$$

The number of cysts administered was 30 for group A and 15 for groups C and D. The percentage of resistance was also calculated for the animals from groups C and D as follows (Christensen et al., 1986):

$$100 - [\text{(average worm burden of experimental group D)} \div \text{(average worm burden of control group C)}] \times 100.$$

The mean dry weight per worm was determined after drying the parasites from all the infected groups at 60°C for 48 hr. At necropsy, the relative splenic and adrenal weights were determined from all the groups as follows (Huffman et al., 1988):

$$\frac{\text{weight of organ (g)}}{\text{weight of hamster (g)}}.$$

Spleens were fixed in 10% neutral buffered formalin, dehydrated through a graded alcohol series, embedded in paraffin, sectioned at 6 μm, and stained with hematoxylin and eosin. For detection of lipids, adrenal glands were embedded at −20°C in O.C.T. (Ames Co., Elkhart, IN), sectioned at 8 μm on a CTF microtome-cryostat (International), and stained with oil red O in propylene glycol.

Student's *t*-test was applied to determine significance between experimental groups ($P < 0.05$).

There was no significant difference in the average litter size between infected hamsters (group A, 8.4 ± 2.7) and uninfected hamsters (group B, 9.4 ± 4.0). The litters from group A did not differ in weight when compared with controls when monitored daily for 2 wk postparturition.

At necropsy, gross pathology was noted for groups A, C, and D. The small intestine of the uninfected controls (Group B) typically had 2–5 enlarged serosal lymphatic nodules, $\bar{x}$ ± SD = 3.6 ± 1.3. Infected animals (group A) had 4.3 ± 1.9 (range, 0–7) lymphatic nodules. All parasites were found in the lower two-thirds of the small intestine.

The percentage of infectivity for groups A, C, and D are given in Table 1. In group C, the animals born from uninfected mothers showed a greater infectivity at ages 6, 7, and 8 wk when